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*Medical Economics* • July through December 1966

Each listing shows title of major article or short item (in italics). First two figures following title indicate date of issue; last figure indicates page number in that issue on which the article or item starts. Back copies of *MEDICAL ECONOMICS* may be purchased, as long as the supply lasts, at \$1 each, postpaid.

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"I suggest, Miss Pomeroy, that you shorten your engagement."

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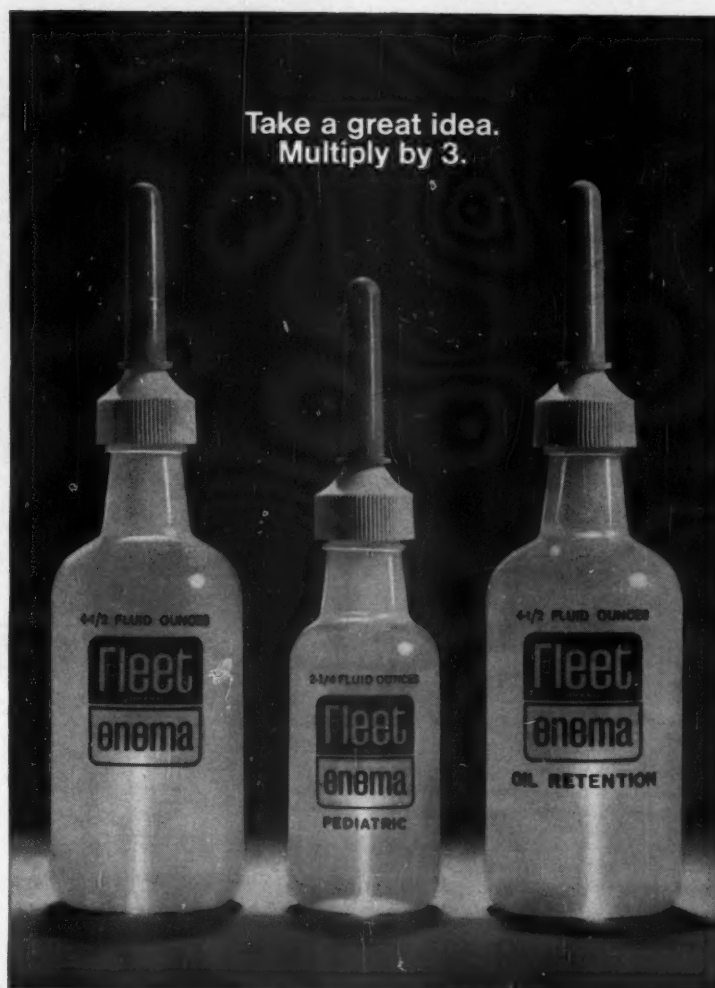
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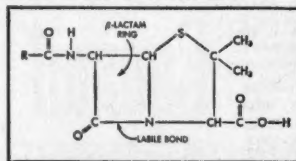
### SQUIBB NOTES ON THERAPY

## Penicillin G and the penicillinase resistant semisynthetics— consider the differences in pharmacologic action, in antibacterial effect

Within the last decade few molecules have been as vigorously investigated as that of penicillin. The result—new semisynthetic penicillins with differences in their actions on bacteria and their interactions with the host. These differences may greatly affect their use in clinical practice.

In the October 16, 1965 issue of the Journal of the Canadian Medical Association, Dr. H. Kalant<sup>1</sup> of the Department of Pharmacology of the University of Toronto discusses the theoretical basis for these differences.

### Destruction of Bacteria



Basic chemical structure of penicillin

The basic structure of penicillin is 6-amino-penicillanic acid.

The important part of the penicillin molecule, as far as antibacterial action is concerned, is the highly reactive and relatively unstable beta-lactam ring.

This is how it may react with bacteria: Bacteria are surrounded by a rigid outer wall which is continually being synthesized from within. The synthesized material is transferred by enzyme action to the outer wall.

When penicillin comes into contact with susceptible bacteria, the labile bond in the C-N linkage of the beta-lactam ring breaks.

The C attaches itself to receptor sites on the transferring enzymes of the bacteria, preventing further synthesis of the outer wall. Without the protection of the outer wall, the bacteria rupture from internal osmotic pressure.

### Penicillin and penicillinase

Apparently the beta-lactam ring is the key to effective antibacterial action. But it is also the key to the inactivation of penicillin.

Certain bacteria (resistant staph, for example) produce the enzyme penicillinase. When penicillinase comes into contact with penicillin it breaks the C-N linkage of the beta-lactam ring. The C reacts with the penicillinase to form penicilloic acid. Since the C is no longer free to react with the bacterial enzymes, there can be no destruction of the bacteria and the enzymatic action of penicillinase remains unchanged.

### Penicillinase is counteracted by certain semisynthetics

Modifications to the R side chain of the molecule alter the reactivity of the penicillin—either by altering the stability of the beta-lactam ring or by protecting the labile C-N bond from hydrolysis by penicillinase.

The newer semisynthetic penicillins protect the C-N bond with bulky R groups arranged to shield it from the action of penicillinase. This process is called steric hindrance.

Some semisynthetic penicillins, when they come into contact with the penicillinase, bind themselves to the penicillinase, altering the penicillinase as they lose their activity.



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## Molecular changes to counteract penicillinase, however, may inhibit action against bacteria

These are some of the changes that may take place when the R side chain of the penicillin molecule is modified:

1. Penicillinase-resistant penicillins have a considerably lower intrinsic activity than penicillin G—because the same steric hindrance (R group arrangement) that shields the C-N bond from penicillinase probably also impairs its ability to react with the bacteria. It could also be due in part to their slower diffusion through the outer cell wall of the bacteria to the inner receptor sites.

2. Modifying the R side chain of penicillin may change the amount of it bound to serum-protein. When protein-binding is high, use of protein-free media for antibiotic susceptibility tests may have little relation to clinical effectiveness.

3. Finally, it was first thought that allergy was due to sensitivity to the basic penicillanic acid nucleus. It is now suggested that sensitivity may be due in part to some degradation product of penicillin—and that more than one type of allergen is involved.

The differences in penicillins, as described by Dr. Kalant in his article, are of significance in selecting antibacterial therapy. There is another statement that he makes, however, that is also important.

"...Penicillin G remains the most active of all penicillins against susceptible bacteria."<sup>1</sup>

## Penicillin G is still the therapy of choice against susceptible organisms

This is the reason so many investigators,<sup>2-8</sup> in both laboratory and clinical studies, have found penicillin G to be the drug of choice against pneumococci, streptococci and sensitive staphylococci.

The new semisynthetic penicillins have a definite place in antibacterial therapy... particularly in the treatment of penicillin-resistant strains of staphylococci.

For oral or parenteral penicillin treatment of infections other than those involving resistant strains of staphylococci, the American Medical Association Council on Drugs, in the 1965 edition of *New Drugs*, states: "Penicillin G is still the most widely

used penicillin. It remains the drug of choice for the treatment of infections caused by susceptible gram-positive cocci such as pneumococci, group A hemolytic streptococci, and sensitive staphylococci, except for patients who are allergic to the penicillins... Penicillin G is also preferred for infections caused by gonococci, clostridia, *Bacillus anthracis*, *Corynebacterium*, *diphtheriae*, and *Actinomyces* species."<sup>5</sup>

**CONTRAINDICATIONS:** Oral penicillin G is not recommended in syphilis, subacute bacterial endocarditis or meningitis; not for persons with hypersensitivity to penicillin.

**PRECAUTIONS AND SIDE EFFECTS:** Reactions to oral penicillin are essentially limited to sensitivity phenomena, and are most likely to occur in individuals with an allergic history or with demonstrated penicillin hypersensitivity. Such reactions may include urticaria, serum sickness-like reactions (fever, rash, arthralgia), other skin rashes, and, rarely, anaphylactoid shock. In case of serious anaphylactoid reactions, epinephrine, oxygen, and intravenous corticosteroids may be required immediately. Observe for possible overgrowth of nonsusceptible organisms. Loose stools may be encountered and an occasional patient may complain of sore mouth or tongue.

**DOSAGE:** 200,000 or 400,000 units t.i.d.

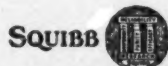
**SUPPLY:** Tablets (crystalline potassium penicillin G buffered with calcium carbonate) 200,000 units in bottles of 16, 100, 500; 400,000 units in bottles of 16, 100; 800,000 units in bottles of 30, 100; Capsules (crystalline potassium penicillin G) 200,000 units, bottles of 100, 500; 400,000 units, bottles of 50; For Syrup (potassium penicillin G with sodium phosphates as buffers) 200,000 units or 400,000 units for 5 cc, teaspoonful, bottles for reconstitution to 80 cc. (16 doses) and 150 cc. (30 doses). For full information, see Product Brief.

**REFERENCES:** 1. Kalant, H. J. Canadian M. A. 93:839, 1965. 2. Friend, D. G.: Clin. Pharm. & Therap. 7:421, 1966. 3. Eickhoff, T. C., and Finland, M.: Am. J. M. Sc. 249:261, 1965. 4. Kislak, J. W., et al.: Am. J. M. Sc. 250:261, 1965. 5. Evaluated by A.M.A. Council on Drugs: New Drugs, 1965 ed., Chicago, American Medical Association, 1965, pp. 9-10. 6. Finland, M.: Med. Times 93:101, 1965. 7. Breese, B. B.; Disney, F. A., and Talpey, W. B.: Am. J. Dis. Child. 107:232, 1964. 8. High, R. H., and Huang, N. N.: Pediat. Clin. N. Amer. 10:745, 1963.

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